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Problems in the Genetics of Human Obesity

Introduction

IT IS A commonplace of observation that the tendency to put on weight runs in families. This is reinforced when seeing relatives at hospital obesity clinics. The similarity of body build is apparent. This can be seen in Figure 1 in which the somatotypes of a group of obese women and their first degree relatives and the somatotype of a contrast group of normal weight women and their relatives can be compared.

However, between these simple observations and any more precise genetical statement of the situation there are relatively few studies and many problems.

The main difficulty for the geneticist lies in deciding what phenotype to study that will represent what is regarded as clinical obesity. One of the earliest workers, Davenport¹ in 1923 wrote a monograph in which he attempted to use an index of body build (height/wt²). Using this index he divided the population into five groups (very slender, slender, medium, fleshy and very fleshy) and classified parents and children on this basis. Apart from this early study, where an awareness of the complexity of the classification of the phenotype was shown, most other workers have assumed that obesity is an all or none condition. Therefore a knowledge of the inci-

dence of obesity among the parents of the obese and a knowledge of the incidence of obesity among the offspring of certain types of mating has been the result of these other inquiries. For instance Bauer² showed that of 1,000 obese patients, 73 per cent had one or both parents obese. In Chicago, Rony³ found that 173 out of 250 patients had one or both parents obese. Other similar studies^{4,5,6,7,8} are in the literature. Gurney⁹ showed that 73 per cent of the offspring of *stout* × *stout* matings were themselves *stout*. Whereas in a *stout* × *not stout* mating only 41 per cent of the offspring were *stout*.

Gurney interpreted this result as showing that stout individuals carry the genes for slenderness and these can segregate in the gametes whereas the slender individuals rarely carry gametes for stoutness and are more likely to be recessive. Angel¹⁰ in a comprehensive study of obese white women in Philadelphia studied the ratio of *fat* : *thin* among the offspring. His results are given in Table 1.

He noticed that *Fat* ♂/*Average* ♀ and *Average*/*Average* matings produced fewer males among the offspring and at the same time larger families. This lead him to the hypothesis that one of the genes helping to determine obesity is a sex-linked recessive gene. He commented that

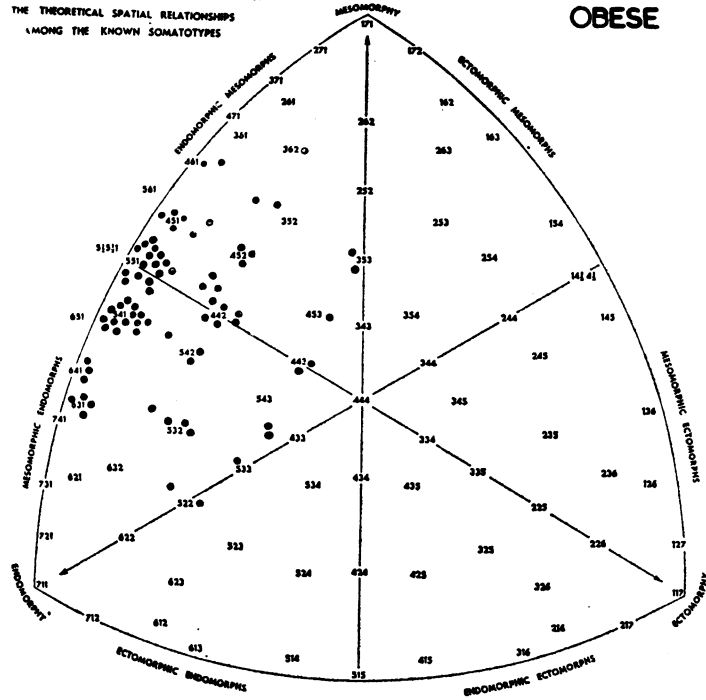
TABLE 1
TYPES OF MATING IN PHILADELPHIA POPULATION OF OBESE WHITE WOMEN
(From Angel.)

TYPE OF MATING	%	OFFSPRING		TYPE OF MATING	<i>Fat</i> / <i>thin</i>	SEX RATIO	
		<i>Fat</i> ♂ ♀	<i>Ave.</i> ♂ ♀			<i>Boys</i> /100 <i>girls</i>	<i>Family</i> <i>size</i>
Fat ♂ × Fat ♀	25.9	32 51	25 22	Fat ♂ × Fat ♀	.70	78.1	4.33
Average ♂ × Fat ♀	36.2	33 69	54 54	Average ♂ × Fat ♀	.49	76.7	5.00
Fat ♂ × Average ♀	15.5	13 27	19 11	Fat ♂ × Average ♀	.57	84.2	3.89
Average ♂ × Average ♀	22.2	8 40	43 37	Average ♂ × Average ♀	.37	66.2	4.92

THE EUGENICS REVIEW

A SCHEMATIC TWO-DIMENSIONAL PROJECTION OF
THE THEORETICAL SPATIAL RELATIONSHIPS
AMONG THE KNOWN SOMATOTYPES

OBESE



A SCHEMATIC TWO-DIMENSIONAL PROJECTION OF
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RELATIVES
OF
OBESE

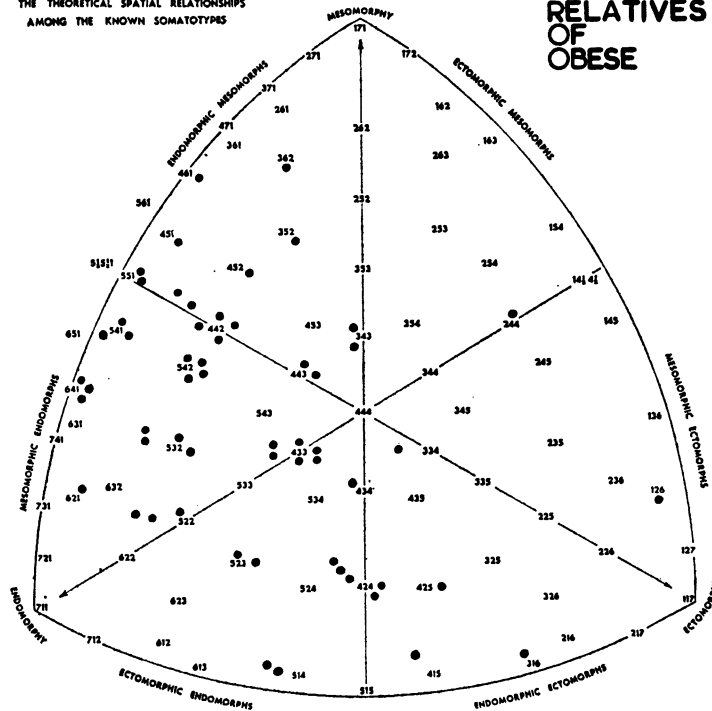
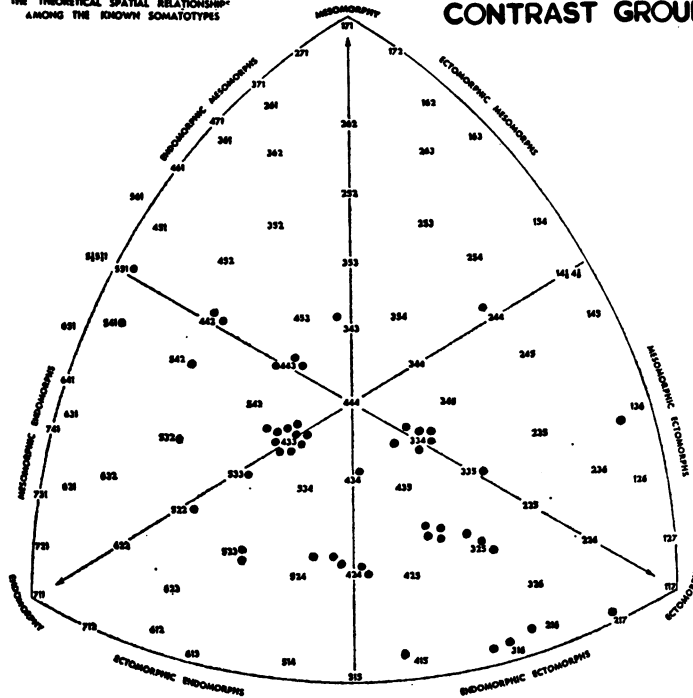


FIGURE 1 COMPARISON OF BODY BUILD OF OBESE POPULATION AND THEIR RELATIVES

PROBLEMS IN THE GENETICS OF HUMAN OBESITY

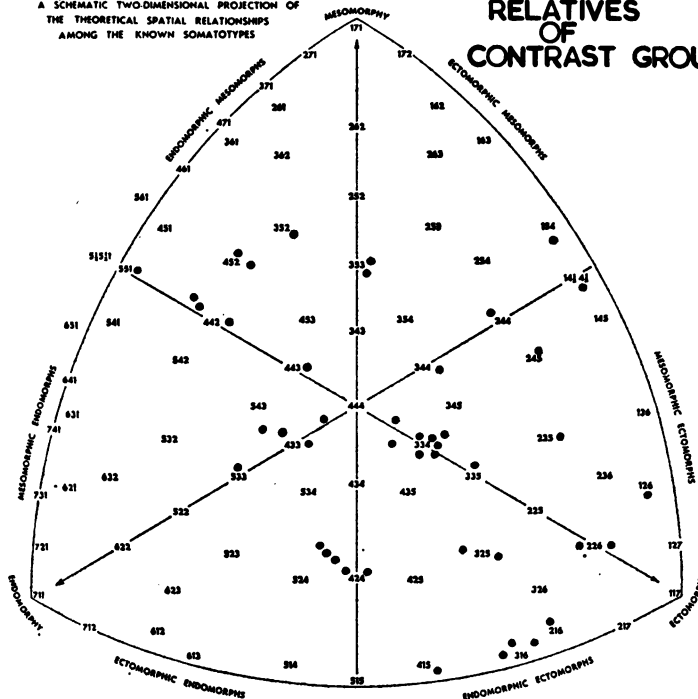
A SCHEMATIC TWO-DIMENSIONAL PROJECTION OF THE THEORETICAL SPATIAL RELATIONSHIPS AMONG THE KNOWN SOMATOTYPES

CONTRAST GROUP



A SCHEMATIC TWO-DIMENSIONAL PROJECTION OF THE THEORETICAL SPATIAL RELATIONSHIPS AMONG THE KNOWN SOMATOTYPES

RELATIVES OF CONTRAST GROUP



AND BODY BUILD OF CONTRAST GROUP AND THEIR RELATIVES

the relatively large family size of the two matings may have a purely psychological or social-biological and not a genetic determination.

In the study which the author has carried out he thought it worth while to prepare similar tables to those of Angel for an obese population and a control population.

The obese population were selected on the basis of the obesity of one of the offspring (see below) and the propositus is left out of the offspring figures in the table. The control population was a population of factory women (see below). Again the propositus was omitted from the table.

The consistency of the *Fat/thin* ratio in *Fat* × *Fat* mating in this British data and the earlier work in the literature is striking. The sex-ratios and family size do not show consistency and are different from Angel's data. This suggests that variable social factors underlie these differences.

Steinberg¹¹ in a recent review describes such approaches as the above as anecdotal. He takes this position because he sees quite correctly that obesity is *not* an all or none clinical condition. He presents a definition of obesity as an excessive accumulation of fat—beyond 10-20 per cent of the normal range for a particular age, height and sex. In presenting this definition Steinberg is emphasizing that obesity involves a continuous variable. In his definition this is represented by *fatness* which varies continuously throughout the population. There are other definitions which regard the obese as those who

are over 20 per cent overweight. Here again a continuous variable is spoken of. Genetically such conditions are believed to be due to a series of genes with additive or multiplicative effect or both, and are believed to vary with certain environmental factors. The phenotype that one studies is a product of a varying multifactorial genetic situation developing in varying environments.

In animal and plant populations such genetic situations can be studied by experimental breeding. The influence of genetic factors in milk yield can be studied by complex breeding and interbreeding within herds. In human populations such techniques are not available. Therefore special devices have to be sought in place of experimental breeding.

A number of possible solutions to this problem were mentioned by Steinberg and they include:

1. *Twin Studies*: Identical (Monozygous) twins can be compared with Dizygous twins and with ordinary siblings. Moreover, if a sample of identical twins reared apart can be studied then some idea of the environmental component can be obtained.

2. *Family Correlations*: If the continuous variable can be corrected for height, age and sex then correlation coefficients could be computed between relatives so that family correlations would show genetic resemblances between the relatives. These would hold on the assumption of random mating, although it is worth pointing out at the beginning that psychological factors respecting body build almost certainly affect the choice of mate.

TABLE 2
RESULT OF DIFFERENT TYPES OF MATING IN AN OBESE POPULATION

TYPE OF MATING	%	OFFSPRING				TYPE OF MATING	<i>Fat/ thin</i>	SEX RATIO	
		<i>Fat</i>	<i>Ave.</i>	<i>Fat</i>	<i>Ave.</i>			<i>Boys/100 girls</i>	<i>Family size</i>
Fat ♂ × Fat ♀	43	♂ 39	♀ 24	♂ 33	♀ 52	Fat ♂ × Fat ♀	·74	87·7	4·2
Average ♂ × Fat ♀	28·5	16	8	21	33	Average ♂ × Fat ♀	·44	155·5	3·4
Fat ♂ × Average ♀	13·2	6	0	14	16	Fat ♂ × Average ♀	·2	139·9	3·26
Average ♂ × Average ♀	15·3	2	5	19	32	Average ♂ × Average ♀	·14	61·9	4

TABLE 3
RESULT OF DIFFERENT TYPES OF MATING IN A CONTROL POPULATION

TYPE OF MATING	%	OFFSPRING				TYPE OF MATING	<i>Fat/ thin</i>	SEX RATIO	
		<i>Fat</i>	<i>Ave.</i>	<i>Fat</i>	<i>Ave.</i>			<i>Boys/100 girls</i>	<i>Family size</i>
Fat ♂ × Fat ♀	18	12	15	18	19	Fat ♂ × Fat ♀	·73	79·6	3·5
Average ♂ × Fat ♀	32·5	11	18	62	69	Average ♂ × Fat ♀	·239	100	4·3
Fat ♂ × Average ♀	20	4	10	31	25	Fat ♂ × Average ♀	·25	103·6	5·2
Average ♂ × Average ♀	29·5	5	16	45	27	Average ♂ × Average ♀	·29	109·8	3·66

3. *Environmental Studies*: Apart from the use of twin studies for examining environmental factors there is still the problem of the magnitude of environmental influences on the correlations between relatives. This could be studied by the use of parent-child correlations in a parent-adoptive population. Here any such correlation must be due either to bias in the selection of the child by the adopting parents (which in Britain is negligible because of our adoption system) or to environmental factors such as family eating patterns. I have used this method as will be seen later. Steinberg is attempting to investigate this by studying a group called the Hutterites—an Anabaptist sect which lives in a fairly stable environment sharing the same food, clothes and living a communal life. So far he has not reported any findings from this study.

The Phenotype

Given a metric of some sort which can be used as synonymous with obesity an attempt can be made to see how much of the variation in this metric can be associated with genetic factors. In so far as the metric must be something which is correctable for height, weight and age, many of the more natural measurements associated with obesity cannot at the moment be used because of a lack of correcting factor. For example, several suggestions have been made in the literature for indices of adiposity. Total body fat could be used, or some anthropometric measure of body fat. Unfortunately at the moment there are no comprehensive tables of standards for such indices available. This is why Steinberg's suggested definition is not helpful at the present time. On the other hand insurance companies find it worth making those people who are overweight pay an increased premium for their obesity. It was therefore decided to take overweight as the phenotypic characteristic. When the question is asked "overweight with respect to what"? Two answers might be given. Firstly, on the basis of table of life expectancy the insurance companies have evolved the concept of "ideal weight". This concept is related both to life expectancy and the amount of profit which such companies make in handling particular mortality risks. It is also worth noting that ideal weights for heights and

ages over thirty years are very much lower than the average weight at that height and age. A person of *average weight* at forty-five years of age would be more than 20 per cent overweight with reference to *ideal weight* at that age. It was decided to use *average weight* based on the table for the British population drawn up by Kemsley¹² when considering adults and the tables of Sutcliffe and Canham¹³ for children. A given individual would be evaluated against these tables and a value—the *percentage overweight* could be assigned to him or her. It should be noticed that this percentage can be positive or negative, and in the latter case the person would in fact be underweight. However, for mathematical simplicity he would be treated as having negative overweight.

The Initial Population

Two populations of working class women living in a south London suburb were studied. The first population consist of 130 consecutive obese patients attending a clinic for obesity at their local hospital. These were compared with 250 women volunteers working at factories in the neighbourhood. It was felt that such a control population would be a sample of women from the population of which the obese group were drawn. It included women of all types of body build. The factory doctor was asked to obtain volunteers for research into what was in the diet which affects health. This form of invitation was used in the hope that the sample would not be unduly biased with respect to weight. The volunteers represented about half the available population at each factory.

When each propositus was seen, whether they were from the obese or control populations it was explained to them that the author was interested in the way in which their eating habits ran through their family and an attempt was made to obtain their permission for all their first degree relatives to be contacted and visited. About fifty of the control population refused this permission. Further information was lost through some of the relatives refusing to co-operate when they were contacted, but this applied only to 23 per cent of the relatives of either group.

When a person was seen they were investigated

in the following ways and in the following order. An inquiry was made into their weight at certain times throughout their life, for example were they plump as a child? What was their weight at marriage? What was their weight at menopause? An account of their pregnancies was elicited for we have found, with Angel, that gain in weight in pregnancy is the commonest given cause of adult obesity in women. (N.B. this is not to say that it is the real cause.) A medical history was taken and this included any account of emotional disturbance to see if there might be any association between such a disturbance and gain in weight. Next dietary history was taken. Their average daily diet was investigated and inquiries made about any food preferences they might have. This was done to consider the possibility that the obese and their relatives might tend to eat more starchy foods than the controls.

Then each person was weighed and somatotyped by Parnell's¹⁴ method. This method was chosen because of the difficulty of photogrammetric somatotyping in a house to house survey. The addresses of each first degree relative was then asked for together with a description of age and build, marital status and family size of each relative. This inquiry produced information about deceased relatives and such things as build was checked against photographs if any were available. In fact when these descriptions were compared with the actual build of those relatives who co-operated and were subsequently seen there was sufficient agreement to feel fair confidence in this hearsay evidence. It is on the basis of this information that the Tables 2 and 3 above were produced.

Finally each person, over the age of sixteen, was asked to complete a personality inventory which has been designed to test for possible depressive psychogenic factors in the etiology of obesity. It is worth noting that this questionnaire appeared to be somewhat disturbing, because we found that some women refused to attend when they heard about the questionnaire from their colleagues so the sample of control women is biased by this factor.

Only a brief account of the results of this whole survey can be given here. Full details will be published elsewhere. The inquiry was carried out in the hope some single factor might

be shown to contribute to the family incidence of obesity, as well as to obtain the data for a multifactorial analysis of the phenotype.

Multifactorial Analysis of Human Overweight

The first findings of the sample of factory workers and the obese population have been published elsewhere.¹⁵

The following correlations were found:

Offspring/parent

Regression coefficient $b = 0.1453 \pm 0.0402$

Correlation coefficient $r_{p/o} = 0.127 \pm 0.0633$

Full Sibs

Intra class correlation coefficient $r_{s/s} = 0.259 \pm 0.0408$

$$\text{Heritability} = 2b = 0.29$$

When these figures are substituted in the usual formulae the partitioning of the variance for dominance and for environmental factors become nonsensically high.

From Mather:¹⁶

$$D (\text{Variation due to fixed genetic factors}) = 2r_{p/o} = 0.254$$

$$\frac{H}{D} = \frac{\text{Unfixable genetic}}{\text{Fixable genetic}} = \frac{4(r_{p/o} - r_{p/o})}{r_{p/o}}$$

$$H = 4.157D (\text{Dominance, etc.})$$

$$1 - 2r_{p/o} - \frac{H}{D} r_{p/o}$$

$$\frac{E}{D} = \frac{\text{Non-heritable}}{\text{Fixable genetic}} = \frac{\frac{H}{D}}{4r_{p/o}}$$

$$E = 0.4293 D (\text{Environmental factors})$$

In reporting these findings I suggested that the fault lies in the model used which was designed for *experimental* breeding among animals and plants. In human genetics environmental factors will lead to what I called *social* inheritance as opposed to genetic inheritance. Family social patterns such as eating habits or the psychological milieu of the family will contribute to parent/offspring correlations and full sib correlations. They will tend to make the within-family variance less than the between-family variance, just as does genetic inheritance. Also in so far as these factors might affect all the siblings they will present to the geneticist as dominance.

A way round this difficulty is to measure such factors by finding the offspring/parent correlations in families which have at least one *adopted*

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child. Since there should be no genetic relationship in such families, any correlations found must measure part of the social inheritance belonging to the family situation.

The ten major adoption societies in this country were contacted but only one, Dr. Barnado's, would co-operate. This Society sent a form to all parents who had adopted children who would be from 8-13 years old. Three hundred forms were sent out, and 142 were returned to me from parents who remained anonymous so far as I was concerned. Forty-two forms were returned as unknown at the address sent.

The parent/offspring correlation coefficient were calculated and can be compared with those obtained for the factory population.

Adoptee population

Father/ natural child	$r_{f/o} = 0.592$	N = 13	Not Significant*
Mother/ natural child	$r_{m/o} = 0.34$	N = 13	Not Significant*
Father/ adoptee	$r_{f/ao} = 0.113$	N = 142	Not Significant*
Mother/ adoptee	$r_{m/ao} = 0.157$	N = 142	

Factory population

Father/ offspring	$r_{f/o} = 0.42$	N = 36	Significant
Mother/ offspring	$r_{m/o} = 0.162$	N = 242	Significant

The significant correlation can be expressed on arrows as in Figure 2.

Such correlations suggested at the time *sex-limitation*. However, the population was small

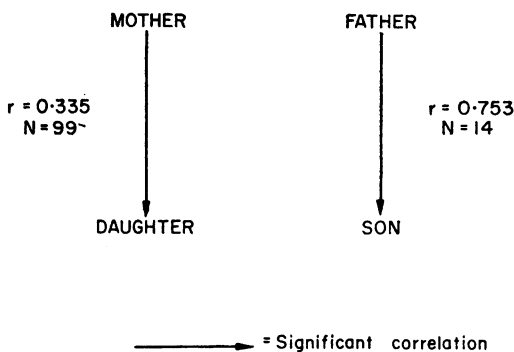


FIGURE 2

FACTORY POPULATION—PARENT/OFFSPRING CORRELATIONS

* This means not significantly different from zero.
N = Number in sample.

owing to the shortage of fathers who made themselves available for interview, and it was decided to seek a larger sample elsewhere. A sample of children aged 10-18 from boys' and girls' schools in a London borough, Watford, was taken. The children were given a form on which they had to fill in their own height, age and weight and those of their first degree relatives. This was presented to them through the schools with an injunction as to necessary accuracy and the research spirit behind the investigation. This yielded a large population whose family correlations were calculated.

Father/son	$r_{f/s} = 0.139$	N = 338	Not Significant
Father/ daughter	$r_{f/d} = 0.2387$	N = 412	Significant
Mother/son	$r_{m/s} = 0.192$	N = 333	Significant
Mother/ daughter	$r_{m/d} = 0.1855$	N = 414	Significant

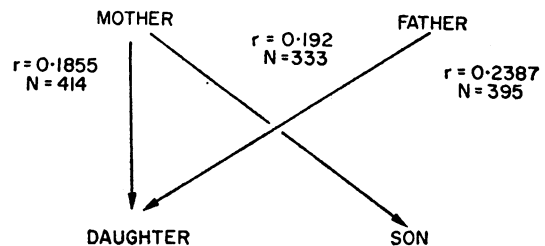


FIGURE 3

WATFORD SCHOOLCHILDREN PARENT/OFFSPRING CORRELATIONS

This pattern of significant correlations suggests that one of the factors is sex-linked. If this is the case, as Mather and Jinks¹⁷ have shown the relation $r_{s/s} > r_{b/b} > r_{s/b}$ should hold, assuming that the effects of the autosomal genes and non-heritable agencies are the same for both sexes.

<i>Theoretical</i>	(Mather)	$r_{s/s} > r_{b/b} > r_{s/b}$	
<i>Observed:</i>			
Sister/sister	$r_{s/s} = 0.2204$	N = 195	Significant
Brother/ brother	$r_{b/b} = 0.2891$	N = 146	Significant
Brother/ sister	$r_{s/b} = 0.2779$	N = 319	Significant

This does not confirm the hypothesis of sex-linkage. It was felt that perhaps because many in this population were children undergoing

their adolescent growth spurt this might upset the comparison of their weight with that of adults. Therefore the correlations were calculated for two sub-group groups.

(a) leaving out those children who were under 15 years of age.

(b) leaving out those children aged from 10-18 years.

TABLE 4
CORRELATIONS FOR TWO AGE GROUPS OF WATFORD SCHOOLCHILDREN

AGE	PARENT/OFFSPRING CORRELATION			
	$r_{t/s}$	$r_{t/d}$	$r_{m/s}$	$r_{m/d}$
(a) Omitting 15 or less	0.1378NS N = 214	0.2583 S N = 239	0.1962NS N = 211	0.2896 S N = 238
(b) Omitting 10-18 year olds	0.0578NS N = 119	0.2868 S N = 112	0.183NS N = 111	0.1868 S N = 122

NS = Not Significantly different from zero. S = Significantly different from zero.

It is interesting that on the restricted sample the mother/son correlation becomes not significantly different from zero. In fact, however, the value is only just not significant so that a sample as large as the total sample might have a significant correlation. It is worth pointing out that in these children the father/son correlation is not

significant and is low, so that the original positive correlation based on a very small sample is possibly fortuitous. This leads us to abandon the hypothesis of sex limitation.

The lack of support, however, for the sex-linkage hypothesis resulting from the sibling correlations requires that a closer examination of the concept of overweight be made. Overweight can be due to a number of factors associated with body build. From the point of view of obesity a clinician means that overweight must be due to an excess of fat. Whereas overweight may be due to an excess of muscle or bone. In somatotype terminology, the clinician is interested in *obesity* as in endomorphy, whereas *overweight* may be the result of a high degree of mesomorphy.

The original sample of factory women and their families had been measured for their somatotype by Parnell's method. An attempt was also made to get some picture of the somatotype of the schoolchildren and their families by giving them a choice of types of body build as a description and asking them to use such descriptions to classify the build of their relatives and them-

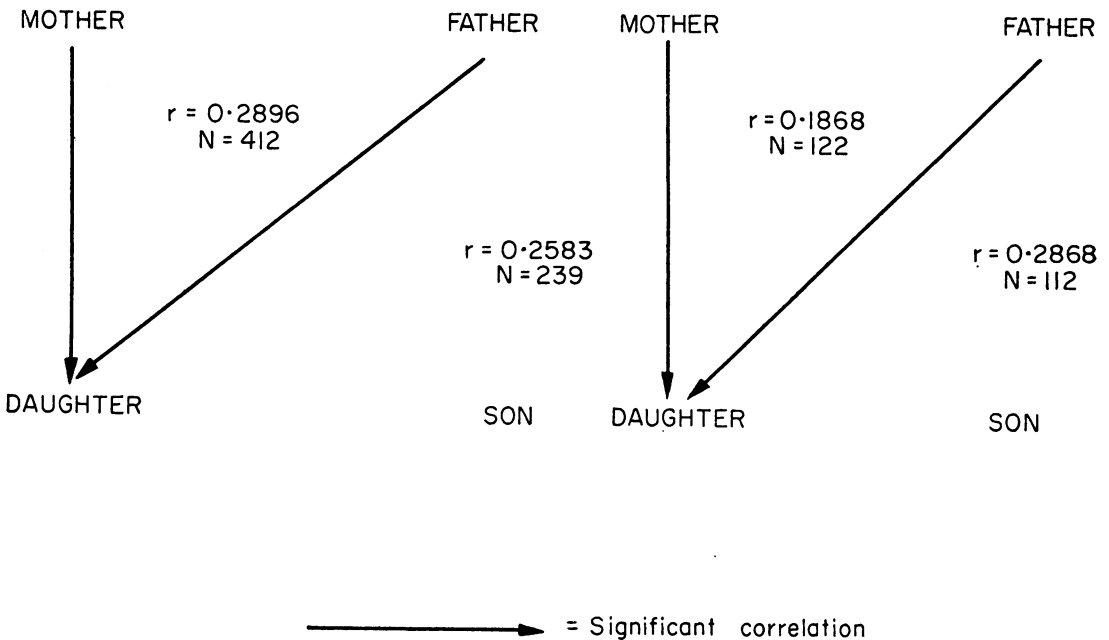


FIGURE 4. WATFORD SCHOOLCHILDREN PARENT/OFFSPRING CORRELATIONS

PROBLEMS IN THE GENETICS OF HUMAN OBESITY

FACTORY POPULATION

WATFORD SCHOOLCHILDREN

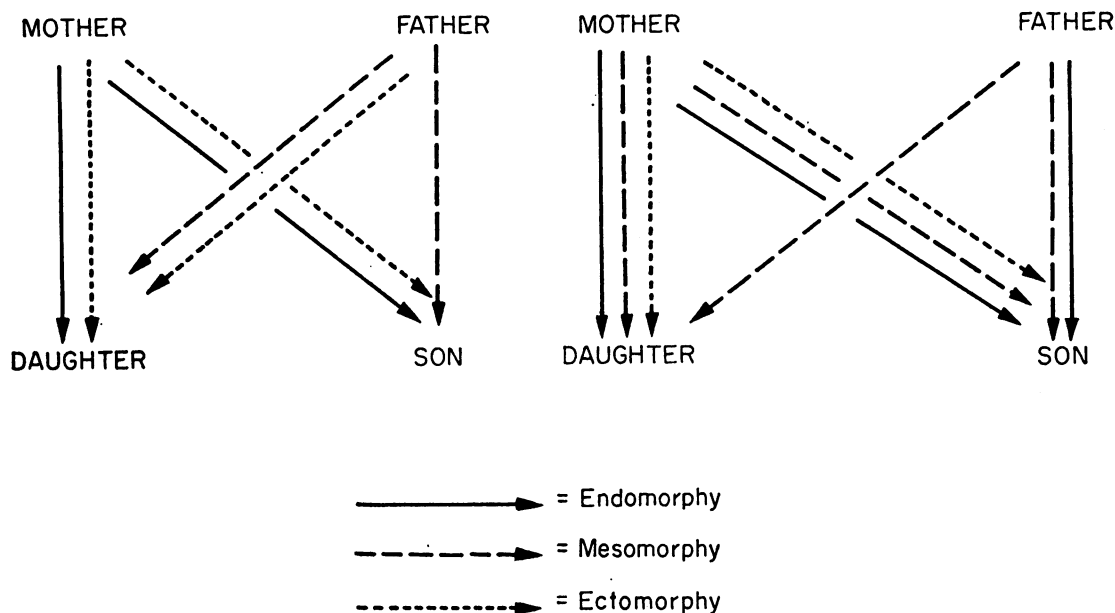


FIGURE 5. SIGNIFICANT CORRELATION FOR BODY BUILD

selves. These descriptions were intended to be related to actual numerical somatotypes, so that these could be calculated. Each component of the somatotype was then correlated in turn between the relatives.

TABLE 5

PARENT/OFFSPRING SOMATOTYPE CORRELATIONS

Results

FATHER/SON	N = 10
Endomorphy	$r = -0.196$ Significant
Mesomorphy	$r = 0.914$ SIGNIFICANT
Ectomorphy	$r = -0.0765$ Not Significant
FATHER/DAUGHTER	N = 14
Endomorphy	$r = 0.268$ Not Significant
Mesomorphy	$r = 0.4618$ Not Significant*
Ectomorphy	$r = 0.524$ Not Significant*
MOTHER/SON	N = 50
Endomorphy	$r = 0.2816$ SIGNIFICANT
Mesomorphy	$r = -0.0523$ Not Significant
Ectomorphy	$r = 0.2409$ Not Significant
MOTHER/DAUGHTER	N = 51
Endomorphy	$r = 0.1825$ Not Significant*
Mesomorphy	$r = -0.0384$ Not Significant
Ectomorphy	$r = 0.1871$ Not Significant*

* = correlation which with a slightly larger sample might become significant. They indicate a tendency towards a significant correlation.

The figures can be seen in a diagram in Figure 5. The populations have been shown separately for one has actually been measured, the schoolchildren are inferred. The latter is open to serious bias including psychological bias which has vitiated other work done on similar lines.

There are differences in these diagrams which need more accurate and larger samples for their clarification, but two things stand out:

- (1) in neither case does the father contribute ectomorphy to the son.
- (2) the sex cross correlations, i.e. mother/son and father/daughter are basically different. The mother contributes at least her *endomorphism* to the son, whereas the father is contributing mesomorphy to the daughter.

From this it can be seen that the cross correlations which we saw for overweight must have a different basis. They must be based on the mesomorphy relationship for the father/daughter correlation, and on the endomorphy relationship for the mother/son correlation. It looks as

though what is needed is some evaluation of the relative contribution of endomorphy and mesomorphy to overweight.

It must be concluded that the use of overweight as a phenotype in the study of the genetics of obesity is unsatisfactory. It might be better to use total body fat or some other measure of endomorphy. However, as was indicated at the beginning, what is lacking is a set of standard tables for such a phenotype which can connect age, sex and height factors, although standards have been published by Tanner¹⁸ for children. Meanwhile, it is always possible that an abnormality of metabolism may be shown to be the fundamental lesion in human obesity, and that the quantitative approach is only forced on the geneticist by that fact that the *expression* of obesity is in term of *body build* which is genetically extremely complicated.

Acknowledgements

The author wishes to thank Dr. A. Kahan of St. James Hospital, Balham for offering valuable facilities and his interest and encouragement. Also the directors of the factories in which the control population was seen for their co-operation. Thanks are also due to Dr. Alcock and Dr. Gough-Thomas who assisted with the Watford schoolchildren. Especially does he wish to thank Mrs. E. V. Leckie who assisted with visiting, performing many of the calcula-

tions of the research and helping in many other aspects of the work. Finally grateful thanks are given to the Eugenics Society for their generous financial help in making this survey possible.

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